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REMARKS

Claims 1-11 and 30-34 are currently pending. Claims 1 and 7 have been amended to more particularly claim the invention. Support for the amendments to claims 1 and 7 can be found throughout the specification, and in particular on page 15, lines 19-21 and page 16, lines 11-14. Applicants respectfully request reconsideration and allowance of all pending claims.

1. Rejection of the claims under 35 U.S.C. §112, First

Paragraph

Reconsideration is requested of the rejection of claims 1-11 and 30-32 under 35 U.S.C. §112, first paragraph as failing to comply with the written description requirement. Specifically, the Office has stated that the specification and claims as originally filed fail to provide adequate written description for the limitation directed to the step of identifying an overweight or obese mammal.

Although applicants disagree with the Office's position for the reasons set forth in the previous response dated July 27, 2009, in order to further prosecution, this phrase has been deleted from independent claims 1 and 7. In light of this amendment, applicants submit that the rejection of claims 1-11 and 30-32 under §112, first paragraph, has been overcome.

2. Rejection of the Claims under 35 U.S.C. §103(a) over Phinney, et al., Visser, et al., and Bren

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Reconsideration is requested of the rejection of claims 1-4, 6, and 30-33 under 35 U.S.C. §103(a) as being unpatentable over Phinney, et al. (WO 03/043570) in view of Visser, et al. ("Elevated C-Reactive Protein Levels in Overweight and Obese Adults", Journal of the American Medical Association, 1999; 282:2131-215) and Bren ("Losing Weight: Start by Counting Calories," FDA Consumer Magazine, Jan-Feb 2002, Pub. No. FDA 04-1303C, p. 1-6).

Claim 1 is directed to a method for decreasing the appetite of an obese or overweight mammal comprising enterally administering at a time prior to or in conjunction with an appetite-impacting stimulus to said mammal an amount of long-chain n-3 polyunsaturated fatty acid effective to decrease the appetite of said mammal, wherein the polyunsaturated fatty acid has 20 or more carbon atoms, and wherein the polyunsaturated fatty acid is administered in the form of a triacylglycerol to treat obesity or overweight in mammals that are obese or overweight, and wherein the appetite of the mammal needs to be decreased and the long-chain n-3 polyunsaturated fatty acid is administered to the mammal for the purpose of decreasing the appetite of the mammal.

Phinney, et al. disclose formulations and methods for the treatment and/or amelioration of symptoms of inflammatory conditions and associated systemic inflammatory responses. Phinney, et al. disclose that elevated levels of C-reactive protein (CRP) have been associated with these various inflammatory conditions. The formulations comprise a non-alpha

tocopherol (especially gamma-, beta-, or delta-tocopherol) and one or more of an omega-3 fatty acid, such as docosahexaenoic acid (DHA) or a flavonoid.

Significantly, Phinney, et al. fail to disclose a method of enterally administering at a time prior to or in conjunction with an appetite-impacting stimulus to said mammal an amount of long-chain n-3 polyunsaturated fatty acid effective to decrease the appetite of said mammal, wherein the appetite of said mammal needs to be decreased. More particularly, **nowhere is there any mention of an appetite-impacting stimulus** in the Phinney, et al. reference. Phinney, et al. furthermore fail to disclose administering an amount of long-chain n-3 polyunsaturated fatty acid effective to decrease the appetite of said mammal for the purpose of decreasing the appetite of the mammal.

Recognizing that the Phinney, et al. reference fails to teach or suggest each and every limitation of Applicants' claimed invention, the Office cites the Visser, et al. reference for combination with Phinney, et al. Specifically, Visser, et al. is cited for its disclosure of using the guideline parameter of body mass index to identify patients that are overweight or obese and have the C-reactive protein biomarker.

Bren generally describes issues arising due to obesity or overweight. Bren indicates that in order to lose weight, one can eat a low calorie, low-fat diet, limit portion size, and increase physical activity.

In order for the Office to show a *prima facie* case of obviousness, M.P.E.P. §2142 requires a clear articulation of the reasons why the claimed invention would have been obvious. Specifically, the Supreme Court in KSR International Co. v. Teleflex Inc., 127 S.Ct. 1727, 82 USPQ2d 1385, 1396 (2007) noted that the burden lies initially with the Office to provide an explicit analysis supporting a rejection under 35 U.S.C. 103. "[R]ejections on obviousness cannot be sustained with mere conclusory statements; instead, there must be some **articulated reasoning** with some **rational underpinning** to support the legal conclusion of obviousness." The Court in KSR International further identified a number of rationales to support a conclusion of obviousness which are consistent with the proper "functional approach" to the determination of obviousness as laid down in Graham v. John Deere Co. (383 U.S. 1, 148 USPQ 459 (1966). Specifically, as previously required by the TSM (teaching, suggestion, motivation) approach to obviousness, one exemplary rationale indicated requires some teaching, suggestion, or motivation in the prior art references that would have led one of ordinary skill to modify/combine the prior art references to arrive at the claimed invention. Specifically, to reject a claim based on this rationale, the Office must articulate the following: (1) a finding that there was some teaching, suggestion, or motivation, either in the references themselves or in the knowledge generally available to one of ordinary skill in the art, to modify the references or to combine reference teachings to arrive at each and every limitation of the claimed invention; (2) a finding that there

was reasonable expectation of success; and (3) whatever additional findings based on the Graham factual inquiries may be necessary, in view of the facts of the case under consideration, to explain a conclusion of obviousness. The Office has failed to meet its burden under number (1) above, as the combined reference teachings fail to teach or suggest each and every limitation of claim 1, and further, there is no apparent reason for one skilled in the art to combine the reference teachings to arrive at each and every limitation. It simply would not have been obvious to one skilled in the art to arrive at Applicants' claimed combinations.

Specifically, as noted above, nowhere in the cited references (or in the knowledge available to one skilled in the art) is there an apparent reason to combine or modify the references to arrive at the claimed limitation of enterally administering **at the time prior to or in conjunction with an appetite-impacting stimulus** to an obese or overweight mammal an amount of long-chain n-3 polyunsaturated fatty acid effective to **decrease the appetite of said mammal**, wherein the appetite of the mammal needs to be decreased and the long-chain n-3 polyunsaturated fatty acid is administered to the mammal **for the purpose of decreasing the appetite of the mammal**. At best, Visser, et al. disclose that individuals who are obese and/or overweight, as defined using the BMI scale were slightly more likely to have elevated CRP levels,¹ and further, Visser, et al.

¹ As taught on page 2133 of Visser, et al., obese men were 2.13 times more likely and obese women 6.21 times more likely to have elevated CRP levels compared with their normal-weight counterparts.

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and Phinney, et al. recognize that elevated CRP levels are related to systemic inflammatory conditions and associated disorders. Further, Phinney, et al. teach administering a composition comprising non-alpha tocopherol and one or more of an omega-3 fatty acid or flavonoid to treat or ameliorate the various inflammatory conditions. Nowhere, however, is there any suggestion that the composition of Phinney, et al. should be administered for the purpose of decreasing the appetite of the mammal, or at a time prior to or in conjunction with an appetite-impacting stimulus which can increase feed intake by the individual, to treat obesity and/or conditions of overweight. These are requirements of Applicants' claimed invention.

In the Response to Arguments section of the current Office action, the Office continues to contend that Phinney, et al. meet the limitation directed to "enterally administering at a time prior to or in conjunction with an appetite-impacting stimulus" because the human body is a dynamic entity and is constantly in a state of growth and change throughout infancy, adolescence or adulthood such that growth of the human body in any of these three stages would necessarily be present at any time the composition was administered. The Office further states that growth periods are considered to be a period of stress on the human body and require proper nutrition and health in order to achieve growth, and thus fall within the scope of the term "appetite-impacting stimulus." Applicants respectfully disagree with the Office's position.

Specifically, as defined in the instant Specification, an "appetite-impacting stimulus" is a stressor or stimulus that has the effect of increasing food intake (i.e., eliciting an appetitive response). Examples provided include irregular meals, sleep deprivation, and parenteral expectations to excel in school and/or sports.² Particularly, it is beneficial to administer the long-chain n-3 polyunsaturated fatty acid prior to or in conjunction with the stimulus so as to decrease the appetite of an obese or overweight mammal at the time of or prior to the stimulus which has the effect of increasing food intake; that is, the long-chain n-3 polyunsaturated fatty acid is to be administered at the time of or prior to when it is most effective.

The Office has once again stated that the "appetite-impacting stimulus" is not limited to the exemplary appetite-impacting stimuli set forth in the specification, and asserted that applicant has not provided any evidence that the Office's interpretation (i.e., that an appetite-impacting stimulus includes growth periods experienced by the human body) is unreasonable and excluded by the claims.

Applicants again respectfully disagree with this assertion, and point out that the response of July 27, 2009 clearly provides applicants' reasoning as to why the Office's interpretation of "appetite-impacting stimulus" is incorrect. Specifically, it is clear from the description in the specification that an "appetite-impacting stimulus" **is not a**

² Specification at page 18, lines 18-30.

general condition which is present in an individual at all times, as suggested by the Office. In support of this, applicants refer to page 18, lines 18-19 of the specification, which indicate that the appetite-impacting stimulus can be an environmental stimulus or stress. Page 15, lines 21-25 of the specification also suggests that the appetite-impacting stimulus can be "intermittent mild stressors, which by inference may elicit an appetitive response." These passages, along with the exemplary appetite-impacting stimuli or stressors provided in the specification clearly indicate that "appetite-impacting stimulus" as used in the claims is not a general condition which is present in an individual at all times.

Applicants further note that if such a stimulus were with an individual continuously, as suggested by the Office, then such an individual would always be hungry and, thus, have a continuous appetite for food. This is clearly not the case, and the Office has provided no evidence to show that an individual has a continuous appetite for food. As such, applicants submit that there is no disclosure or suggestion in either Phinney, et al., Vissier, et al., or Bren of administering the formulation of Phinney, et al. to an obese or overweight mammal "at a time prior to or in conjunction with an appetite-impacting stimulus."

Additionally, as noted above, there is no mention anywhere in Phinney, et al., Visser, et al., or Bren of using the formulations disclosed in Phinney, et al. for the purpose of decreasing the appetite of overweight or obese mammals, wherein the appetite of the mammal needs to be decreased. Rather, the

formulations and methods of Phinney, et al. are for the treatment and/or amelioration of symptoms of inflammatory conditions (e.g., conditions which are associated with an elevated level of C-reactive protein). While Visser, et al. recognize that overweight or obese people may also have the C-reactive protein biomarker, there is nothing in these references (alone, or in combination) to teach or suggest that administering the formulation of Phinney, et al. will affect the appetite of obese or overweight mammals whose appetite needs to be decreased, or would otherwise be effective in the treatment of obesity or overweight conditions. Nor is there any suggestion in any of the cited references that decreasing levels of C-reactive protein would affect the appetite of an obese or overweight mammal.

In this regard, the Office has stated that the suggestion in Phinney, et al. to use the formulations described therein for treating patients exhibiting high levels of C-reactive protein and conditions that are characterized by elevation of C-reactive protein is a clear suggestion to use the formulations in any subpopulation of patients with elevated C-reactive protein, such as those patients suffering from obesity. The Office concludes that the practice of administering the non-alpha tocopherol-DHA therapy of Phinney, et al. for the general purpose of reducing C-reactive protein to treat conditions characterized by elevation of C-reactive protein would also circumscribe its practice in an obese patient population that also exhibits elevated C-reactive protein, and thus meets the instantly claimed method steps. In further support of its position, the

Office has cited Bren. Specifically, the Office takes the position that the teaching in Bren that obesity or overweight can be addressed by reducing calorie consumption supports the notion that the obese individuals in Visser, et al. are mammals who need a decrease or reduction in appetite to maintain a healthy and/or normal weight.

As discussed above, claim 1 requires the appetite of the mammal be one which needs to be decreased. However, not all obese or overweight people have an appetite that needs to be decreased. The Office has in fact agreed with this, stating on page 10 of the April 27, 2009 Office action that "it may very well be agreed that not all obese or overweight mammals have an appetite that needs to be decreased (e.g., such as a patient with a genetic abnormality that causes the obesity and not due simply to overeating)." Thus, the Office is in agreement that not all obese or overweight mammals have an appetite that needs to be decreased. Since, as noted above, there is no disclosure in any of the cited references that the formulations of Phinney, et al. would be effective to decrease appetite, applicants submit that one skilled in the art would not be motivated to modify the method of Phinney, et al. to administer the formulations described therein at a time prior to or in conjunction with an appetite-impacting stimulus to decrease appetite of an obese or overweight mammal whose appetite needs to be decreased, as required in the method of Applicants' amended claim 1.

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The Office has, however, indicated that even though not all obese or overweight mammals have an appetite that needs to be decreased, Bren provides evidence that of all obese and/or overweight mammals, there is a subpopulation therein that is in need of a reduction in appetite to control the obese and/or overweight condition.

The Office appears to be taking the position that overweight or obese mammals in need of a decrease in appetite is a subgenus of the larger genus of obese or overweight mammals, or more generally, mammals exhibiting elevated levels of C-reactive protein, and that since Phinney, et al. use the formulations described therein for treating patients exhibiting elevated levels of C-reactive protein and conditions that are characterized by elevation of C-reactive protein, this is tantamount to a teaching of the use the formulations in any subpopulation of patients with elevated C-reactive protein.

Applicants note, however, that MPEP §2144.08, which deals with the obviousness of a species when the prior art teaches a genus, states: "The fact that a claimed species or subgenus is encompassed by a prior art genus is not sufficient by itself to establish a *prima facie* case of obviousness." Rather, the patentability of a claim to a specific compound or subgenus embraced by a prior art genus should be analyzed no differently than any other claim for purposes of 35 USC 103. In the instant case, for the reasons set forth above, applicants submit that there is no disclosure or suggestion in any of the cited references of enterally administering at the time prior to or in

conjunction with an appetite-impacting stimulus to an obese or overweight mammal an amount of long-chain n-3 polyunsaturated fatty acid effective to decrease the **appetite** of said mammal and treat the obesity or conditions of overweight, wherein the **appetite** of the mammal needs to be decreased, and the long-chain n-3 polyunsaturated fatty acid is administered to the mammal for the purpose of decreasing the appetite of the mammal.

In the Response to Arguments section of the instant action, the Office has also taken the position that since products of identical composition cannot have mutually exclusive properties when administered under identical conditions, whatever effect the long-chain n-3 polyunsaturated fatty acid has in decreasing the appetite of an obese or overweight mammal must reasonably be necessarily present in the method disclosed by Phinney, et al. in view of Visser, et al. The Office thus appears to be taking the position that the method of Phinney, et al. will inherently decrease the appetite of an obese or overweight mammal.

Applicants note, however, that amended claim 1 requires the long-chain n-3 polyunsaturated fatty acid be administered to the mammal for the purpose of decreasing the appetite of the mammal. Although the formulations and methods of Phinney, et al. are disclosed as useful for the treatment and/or amelioration of symptoms of inflammatory conditions (e.g., conditions which are associated with an elevated level of C-reactive protein), and Visser, et al. recognize that overweight or obese people may also have the C-reactive protein biomarker, there is nothing in these references (alone, or in combination) to teach or suggest

that administering the formulation of Phinney, et al. will affect the appetite of obese or overweight mammals whose appetite needs to be decreased, or that treating and/or ameliorating symptoms of inflammatory conditions (including conditions associated with C-reactive protein) would affect the appetite of the subject being treated.

Accordingly, there is no articulated reason to combine or modify the teachings of the cited references to arrive at each and every limitation of Applicants' claim 1. As such, claim 1 cannot be said to be obvious in view of the cited references.

As claims 2-4, 6, and 30-32 depend directly or indirectly from claim 1, claims 2-4, 6, and 30-32 are patentable for the same reasons as claim 1.

Claim 33 is similar to claim 1, except does not require the limitation that the long-chain n-3 polyunsaturated fatty acid is administered to the mammal for the purpose of decreasing the appetite of the mammal. Claim 33 is thus patentable over the cited references for similar reasons as set forth above for claim 1.

3. Rejection of the Claims under 35 U.S.C. §103(a) over
Phinney, et al., Visser, et al., Bogentoft, The Merck Index, and
Bren

Reconsideration is requested of the rejection of claims 7-9, 11, and 34 under 35 U.S.C. §103(a) as being unpatentable over Phinney, et al. in view of Visser, et al., Bogentoft (WO

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87/03198) in further view of The Merck Index (Monograph 972, page 121), and Bren.

Claim 7 is directed to a method for decreasing the appetite of an overweight or obese mammal comprising enterally administering at a time prior to or in conjunction with an appetite-impacting stimulus to the mammal an amount of long-chain n-3 polyunsaturated fatty acid and an amount of long-chain n-6 polyunsaturated fatty acid in amounts effective to decrease the appetite of said mammal, wherein the polyunsaturated fatty acids independently have 20 or more carbon atoms, and wherein the polyunsaturated fatty acids are administered in the form of a triacylglycerol to treat obesity or overweight in mammals that are obese or overweight, and wherein the appetite of the mammal needs to be decreased and the long-chain n-3 polyunsaturated fatty acid and the long-chain n-6 polyunsaturated fatty acid are administered to the mammal for the purpose of decreasing the appetite of the mammal.

For the reasons discussed above, none of Phinney, et al., Visser, et al., or Bren, alone or in combination, teach or suggest each and every limitation of the claimed invention, and further, there is no apparent reason for combining the reference teachings. Bogentoft and the Merck Index fail to overcome these shortcomings. Particularly, there is simply no reason to modify or combine the references to arrive at each and every limitation of claim 7.

Bogentoft discloses enteric preparations in the forms of capsules, tablets, and microcapsules having an enteric coating resistant to gastric juices that dissolves only in the ileum. These enteric preparations contain a hydrophobic substance in combination with an emulsifier. The hydrophobic substance is thus delivered to the ileum, at which point it interacts with specific ileum receptors to induce satiety (page 1, paragraph 3). The enteric preparation is orally administered in a weight reducing dosage to a human. The hydrophobic substance can be a fatty acid having 6-28 carbon atoms, an ester or a salt thereof, a fatty alcohol having 6-28 carbon atoms or an ester thereof.

The Merck Index discloses the formula and properties for arachidonic acid (AA). Specifically, The Merck Index discloses that AA can occur in depot fats of animals.

Similar to Phinney, et al., Visser, et al., and Bren, discussed above, Bogentoft and The Merck Index fail to teach or suggest administering a composition with a long-chain n-3 polyunsaturated fatty acid (as recited in claim 7) and long-chain n-6 polyunsaturated fatty acid prior to or in conjunction with an appetite-impacting stimulus. At best, Bogentoft teach administering their compositions 2-5 hours prior to meal time such that the composition has time to interact with specific ileum receptors to induce satiety to decrease appetite. Nowhere, however, is it taught or suggested that a long-chain n-3 polyunsaturated fatty acid (as recited in claim 7) and long-chain n-6 polyunsaturated fatty acid should be administered prior to a stressor or stimuli that can lead to increased food

intake (i.e., appetite-impacting stimulus), such as sleep deprivation and irregular meal times, such as disclosed in Applicants' specification and claimed in claim 7. The composition of Bogentoft is designed to be used in a completely different manner to treat obesity and conditions of overweight. Thus, the combination of Phinney, Visser, et al., Bren, Bogentoft, and The Merck Index fail to disclose or suggest administering an amount of a long-chain n-3 polyunsaturated fatty acid, as set forth in claim 7, and an amount of a long-chain n-6 polyunsaturated fatty acid to an obese or overweight mammal at a time prior to or in conjunction with an appetite-impacting stimulus, as required by claim 7.

In fact, if anything, Bogentoft actually teaches away from applicants' claimed method. The Supreme Court affirmed in *KSR International Co. v. Teleflex Inc.*, 127 S.Ct. 1727, 1740 (2007), 82 USPQ2d 1385, the holding of *United States v. Adams*, 383 U.S. 39, 51-52 (1996), stating:

The Court relied on the corollary principle that when the prior art teaches away from combining certain known elements, discovery of a successful means of combining them is more likely to be nonobvious.

In the instant case, Bogentoft actually teaches away from decreasing the appetite of an overweight or obese mammal by enterally administering an amount of a long-chain n-3 polyunsaturated fatty acid, as set forth in claim 7, and an amount of a long-chain n-6 polyunsaturated fatty acid, as required by applicants' claim 7. In particular, Bogentoft states:

It has now surprisingly turned out that it is possible to effect a reduced food intake by bringing unabsorbed food and especially hydrophobic [sic] substances therein into contact with the distal part of the small intestine, that is ileum, where a physiologically mediated mechanism having this effect is started. Tests have shown that ileal infusion of a fat emulsion in connection with a meal brings about that a smaller amount of food is ingested than what should otherwise be the case.³

By stating that the method disclosed therein results in a smaller amount of food being ingested "than what should otherwise be the case," Bogentoft suggests that methods of ingestion other than the delivery of fat directly to the ileum would not result in the food intake reduction that is seen with Bogentoft's method.

Additionally, use of the term "otherwise" by Bogentoft implies regular oral administration, not direct delivery of fat to the ileum via enteric coated formulations, as used in Bogentoft's method.⁴ In other words, Bogentoft implies that a similar ingestion of fat by regular administration will not be useful in reducing food intake, but rather that one would need to deliver the fat directly to the ileum to achieve this effect.

³ See Bogentoft at p. 2, lines 18-25 (emphasis added).

⁴ As discussed above, Bogentoft indicate that the enteric preparations disclosed therein are "coated with a coating resistant to gastric juice which dissolves in ileum, the distal portion of the small intestine." Bogentoft at p. 1, first paragraph. The Bogentoft enteral preparations are thus specifically designed for delivery of fat directly to the ileum. In other words, since the coating on the enteral preparations is resistant to gastric juice, the fat is not released until the enteral preparation reaches the ileum. According to Bogentoft, this results in reduced food intake, as explained in the above-quoted passage from page 2 of Bogentoft.

To the contrary, however, applicants have shown that regular administration of "fat" (i.e., enteral administration other than by direct delivery to the ileum), in the form of long-chain n-3 and n-6 polyunsaturated fatty acids, can be useful in decreasing appetite of overweight or obese mammals, provided that the polyunsaturated fatty acids independently have 20 or more carbon atoms and are administered in the form of a triacylglycerol. Thus, applicants have demonstrated that by selecting which fatty acids to deliver, the need for direct ileum delivery of fat to achieve the desired effect of appetite reduction, such as disclosed in Bogentoft, is negated.

Additionally, applicants submit that it would not have been obvious for one skilled in the art to combine the formulations of Phinney, et al. with the fatty acids disclosed in Bogentoft. Although the formulations of Phinney, et al. and the fatty acids of Bogentoft may both be administered to obese or overweight people, these references are concerned with entirely different problems. Specifically, Phinney, et al. teach their composition to have efficacy in the treatment and/or amelioration of symptoms of inflammatory conditions, e.g., by reducing elevated levels of C-reactive protein in patients that suffer from such elevated levels. In contrast, the fatty acids of Bogentoft are used in an enteric preparation for the treatment of obesity, for example, to reduce weight. None of the cited references suggest that the fatty acids of Bogentoft would be effective in treating or ameliorating the symptoms of inflammatory conditions. Likewise, none of the cited references suggest that the formulations of Phinney, et al. would be effective in treating

obesity or overweight by decreasing the appetite of an obese or overweight mammal. Why then would one skilled in the art be motivated to administer to a mammal a formulation for treating or ameliorating inflammation in combination with a fatty acid which is used to reduce weight? There is simply no apparent reason to make such a combination. With all due respect, it appears that the Office has merely used hindsight reasoning in combining these references, which has been specifically instructed against by the Federal Circuit.

Furthermore, applicants note that the only one of the cited references which discloses methods for treating obesity is Bogentoft. As further discussed in the Amendment and Response After RCE submitted August 21, 2008, while Bogentoft state that its hydrophobic substance can be a fatty acid having 6-28 carbon atoms, Bogentoft actually only disclose and enable fatty acids having up to 18 carbon atoms. The fatty acid can be saturated or unsaturated, and have a branched or a straight chain. The fatty acids include lauric acid, palmitic acid, stearic acid, oleic acid, ricinoleic acid, linoleic acid, and linolenic acid. Accordingly, Bogentoft fails to disclose administering an amount of long chain n-3 polyunsaturated fatty acid, wherein the long chain n-3 polyunsaturated fatty acid has 20 or more carbon atoms, effective in decreasing the appetite of an obese or overweight mammal. Specifically, as described in the instant specification, and as required in amended claim 7, "long chain n-3 polyunsaturated fatty acid" refers to fatty acids having 20 or more carbons and having a double bond at the third carbon (see Specification at page 16, line 29 through page 17, line 7).

Furthermore, the only omega-3 fatty acids listed as suitable for the composition of Phinney, et al. include long chain n-3 polyunsaturated fatty acids such as docosahexaenoic acid, having 22 carbons, and eicosapentaenoic acid, having 20 carbons.

As nowhere is it taught or suggested in Bogentoft to administer a long chain n-3 polyunsaturated fatty acid having 20 or more carbon atoms as its hydrophobic substance to be used in the enteric preparation administered for weight loss, Applicants respectfully assert that there is simply no reason for combining the composition of Bogentoft with that of Phinney, et al. In other words, there is no apparent reason to administer long-chain n-3 polyunsaturated fatty acid having 20 or more carbon atoms to a mammal at a time prior to or in conjunction with an appetite-impacting stimulus to decrease the appetite of the mammal. Furthermore, why would one skilled in the art combine the compositions of Bogentoft and Phinney, et al., when each provides ample examples of fatty acids for use in their respective compositions, particularly, when the composition of Phinney, et al. already provides satisfactory fatty acids, all of which have 20 or more carbon atoms, for treating elevated CRP levels (which may or may not include individuals that are obese or overweight). There simply is no apparent reason to do so.

In the Response to Arguments section of the current action, the Office states that applicants' comments regarding the failure of Bogentoft to teach the administration of long chain n-3 polyunsaturated fatty acids with 20 or more carbon atoms is unpersuasive because Bogentoft was not cited for a teaching of

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long-chain n-3 polyunsaturated fatty acids, as this element of the claims is addressed by Phinney, et al. In response, Applicants note that Bogentoft's failure to teach long chain n-3 polyunsaturated fatty acids with 20 or more carbon atoms is noted as further indication that there is no apparent reason to combine the teachings of Bogentoft and Phinney, et al.

As the cited references fail to provide an apparent reason for one skilled in the art to modify or combine the cited references to arrive at the method of amended claim 7, amended claim 7 is patentable over the combination of Phinney, et al., Visser, et al, Bren, Bogentoft, and The Merck Index.

Claims 8-9 and 11 depend directly or indirectly from claim 7. As such, claims 8-9 and 11 are patentable over the cited references for the same reasons as claim 7 set forth above, as well as for the additional elements they require.

Claim 34 is similar to claim 7, except does not require the limitation that the long-chain n-3 polyunsaturated fatty acid and the long-chain n-6 polyunsaturated fatty acid are administered to the mammal for the purpose of decreasing the appetite of the mammal. As such, claim 34 is patentable over the cited references for similar reasons as set forth above for claim 7.

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CONCLUSION

In light of the foregoing, Applicants request withdrawal of the rejections of claims 1-11 and 30-34 and allowance of all pending claims. The Commissioner is hereby authorized to charge any government fees which may be required to Deposit Account No. 01-0025.

Respectfully Submitted,

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